assessed, and if so whether similar findings were noted by them? We also wonder whether other workers have noted variations in tension levels, depending on who is seen to be 'at fault'.

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Psychiatric morbidity in adults with hypopituitarism

We read the recent article by Lynch *et al.* (August 1994 *JRSM*, pp 445–7) examining the potential relationship betwixt growth hormone deficiency (GHD) and psychiatric illness with interest. While somewhat removed from the realm of clinical utility at present, the possible interplay of these factors is of pertinence to all physicians caring for patients with emotional disorders. Upon review of the article, however, it seems as though several important potential threats to validity were not taken into consideration by the authors.

First, all of the patients who had GHD caused by a tumour (as many as 37 of the 40 enrolled subjects) had the tumour treated in some fashion, either medically, surgically, or with radiotherapy; all of which could conceivably affect brain physiology and result in psychiatric illness. Furthermore, might there be a difference in the ability to cope, both emotionally and psychologically, between people who have been told they have tumours and people with other types of chronic diseases? Could there be an increase in depressive symptoms stemming from the release of unknown psychoactive substances from the tumours themselves? In addition, the GHD subjects described had all recently participated in a 'trial of growth hormone replacement'. Is it possible that the patients may have been emotionally affected by a less-than-miraculous outcome of the experiment, or affected in unknown ways by the pharmacologic replacement of other deficient hormones, such as thyroxine, glucocorticoids, testosterone or estrogen? Finally, the assumption in this study that the test and control groups represent two distinct populations may be inappropriate. Alzaid, *et al.*[1] reported that two-thirds of diabetics who underwent insulin tolerance testing at the Mayo Clinic were also found to be growth hormone deficient. Was similar testing done to rule out GHD in the diabetic control group in this study?

Laying these potential confounders aside, the cross-sectional design chosen by the authors is inadequate to support the suggestion of causation made by the authors in the discussion. A claim of causation would be better supported by a longitudinal study showing psychiatric improvement before and after growth hormone replacement in previously deficient subjects. The simple demonstration of association is insufficient to prove any causal relationship.

We appreciate the opportunity to review such a thought-provoking article. Although we may not agree that all of the conclusions drawn in this study are valid, we do agree with the authors that depression in patients with chronic illness is important to discuss and investigate.

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A comparison of the attitudes shown by general practitioners, hospital doctors and medical students towards alternative medicine

I was very surprised about the finding in the article by Mr M R Perkins and his colleagues (September 1994 *JRSM*, pp 523–5) that 93% of general practitioners had, on at least one occasion, suggested a referral for alternative treatment. I have never referred a patient from my general practice for alternative medicine.

Organic pathology cannot be treated by these methods—with the exception of manipulation of a musculo-skeletal lesion by an osteopath. I would refer such patients to an orthopaedic physician or another medically trained specialist.

Only 4 weeks ago a patient with severe back pain asked me about referral to an osteopath. Instead he was referred to an orthopaedic consultant. The X-ray showed him to have metastatic carcinoma in his spine; further investigations revealed the primary to be in the prostate. Radiotherapy has been a dramatic success in relieving the pain and associated disability.

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Diagnosis and management of arteritis

Dr Fisken states (November 1994 *JRSM*, p 719) that I am simply wrong in my assertion that the measurement of the erythrocyte sedimentation rate (ESR) or plasma viscosity as a guide to dose reduction of steroids is well established. Dr Fisken further writes that attempts to monitor the progress of giant cell arteritis by these means will almost invariably end up by overtreating the patient and precipitating iatrogenic Cushing's syndrome and osteoporosis, etc.

He writes that 'the rate of dose reduction should be judged by the clinical state and not by the ESR'. If he will now re-read my letter he will find that he has misquoted me. What I wrote was that once a patient is diagnosed as having giant cell arteritis, prednisolone must be given 80 mg in divided doses every 24 h and that this should be continued until the clinical manifestations have completely resolved and the ESR and/or plasma viscosity has fallen considerably.

I would point out to Dr Fisken that in those patients who present with sudden loss of vision in one or both eyes this may also occur if they have been inadequately treated or if steroid therapy has been stopped too early. In some 30% of patients with giant cell arteritis the presenting symptom is that of blindness in one or both eyes, due to obliteration of the central retinal artery, or more commonly, of the ciliary arteries resulting in ischaemic optic neuropathy.

I have stated that if a patient around the age of 60 or more presents with sudden loss of vision he should be immediately given an intravenous injection of 10 mg dexamethazone before admission to hospital and before the diagnosis has been confirmed. Such patients may have no previous history of such classical symptoms as intractable headache, particularly occipital and temporal, pain in the face, jaw and mouth exacerbated by chewing and tenderness over the scalp and temporal arteries. This, I regard, as an acute medical emergency.

Once on a low maintenance dose of steroids, patients, should be told that any